EXHIBIT I

ADDENDUM REPORT OF JOHN D SCHELL, PH.D.

OVERVIEW OF COMMENTS BY FEDERAL, STATE AND INTERNATIONAL HEALTH AND ENVIRONMENTAL ORGANIZATIONS ON SELECTED CONSTITUENTS IN THE SPOKANE RIVER

CITY OF SPOKANE
V.
MONSANTO COMPANY, ET AL.

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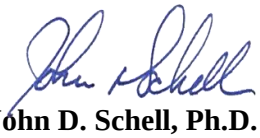

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1.0 INTRODUCTION

The following sections provide brief summaries of the toxicity of selected constituents and bacteria reported by the State of Washington as being present in the Spokane River and that are considered by the state to have the potential to induce adverse impacts to human health and the environment. The summary information is taken from reports by federal, state, and international health and environmental agencies, including the United States (U.S.) Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR); the U.S. Environmental Protection Agency (EPA); the Washington State Department of Health (WDOH), the Centers for Disease Control and Prevention (CDC); the U.S. Geological Survey (USGS); and the European Food Safety Authority (EFSA).

2.0 POLYBROMINATED DIPHENYL ETHERS

Description

Polybrominated Diphenyl Ethers (PBDEs) are a group of man-made organic chemicals that are comprised of monoBDE through decaBDE, with 1 to 46 specific chemical configurations, individually known as congeners, represented within each group (ATSDR 2017). The three predominant commercial PBDE mixtures in the U.S. were pentaBDE, octaBDE, and decaBDE, with each being composed of the stated congener group plus some amounts from other groups (for example, commercially produced octaBDE also contained amounts of penta-, hexa-, hepta-, nona- and decaBDE) (EPA 2008a-d). Over the period from 2004 to 2013, the various PBDEs have been voluntarily withdrawn by U.S. manufacturers (Federal Register 2012). The continued presence of old consumer products and waste materials containing PBDEs and the persistence of the compounds in the environment (particularly in soils/dust and sediments) provide an ongoing source of exposure (ATSDR 2017).

Exposure

People are exposed to PBDEs by inhalation of contaminated air, dermal contact with contaminated soil/dust or consumer products, and ingestion of contaminated foods or soil/dust (ATSDR 2017). The primary route of exposure to PBDEs (accounting for 80% to 90% of the total) for most people is ingestion and (to a lesser degree) dermal contact with contaminated indoor dust, in their homes and places of work (ATSDR 2017). The remaining exposure for most people is from ingestion of contaminated foods, such as fish, meat, and dairy products, which have been shown to contain low concentrations of PBDEs (ATSDR 2017).

Non-Cancer Toxicity

Rats and mice that ingested PBDEs during early development showed neurobehavioral changes and damage to their reproductive systems as adults, with decaBDE exhibiting lower toxicity than the lower-brominated PBDEs (ATSDR 2017). Human studies also suggest an association between PBDE exposure and neurodevelopmental effects; however, limitations of these studies make the results inconclusive. For animal studies (ingestion exposure) with lower-brominated PBDEs administered over several weeks or months, effects to the male reproductive system, thyroid, liver, pancreas (diabetes), nervous system, and immune system have been reported (ATSDR 2017; EPA 2008a-d).

EPA has selected "principal" studies (those which fit specific quality criteria required for derivation of the toxicity values) for four specific PBDEs (BDE-47, BDE-99, BDE-153, and BDE-209) and for two representative commercial PBDE mixtures (octaBDE and pentaBDE). The principal studies for BDE-47, BDE-99, BDE-153, and BDE-209 reported neurobehavioral changes in mice following neonatal exposure (single oral dose, by gavage) (EPA 2008a-d). The principle study for the EPA analyses for octaBDE and pentaBDE reported liver effects (induction of liver enzymes and histologic liver abnormalities) in male Sprague-Dawley rats orally administered commercial-grade octaBDE or pentaBDE (EPA 2019a,b).

Carcinogenicity

According to ATSDR (2017) it is not known if PBDEs can cause cancer in people; however, liver tumors were reported in rats and mice that were fed extremely high levels of decaBDE. No cancer studies have been performed for the other (lower-brominated) PBDEs.

The following carcinogenicity information is based on EPA (2008a-d) and EPA (2019a,b) Integrated Risk Information System (IRIS) information: EPA has classified BDE-47, -99, and -153 as "inadequate information to assess the carcinogenic potential" (EPA 2008a-c), and octa- and pentaBDE "not classifiable" as to human carcinogenicity (EPA 2019a,b), due to a lack of human and animal study data relevant to carcinogenicity. EPA has classified BDE-209 as a "possible human carcinogen" based on the results of cancer studies in animals (EPA 2008d). The principal study used in the EPA carcinogenicity analysis reported liver neoplastic nodules or carcinoma (combined) in male and female rats exposed to decaBDE (BDE-209) in the diet (EPA 2008d).

3.0 MERCURY – METHYLMERCURY

Description

Mercury (chemical symbol, Hg) occurs naturally in the environment and can exist in three forms: elemental or metallic mercury, inorganic mercury (which encompasses numerous mercury salts), and organic mercury (primarily methylmercury) (ATSDR 1999). Metallic mercury is a liquid at normal temperature and is somewhat volatile, particularly when heated. Most-to-all of the mercury found in the tissues of fish is in the form of methylmercury; therefore, that is the form examined in this review (EFSA 2012).

Exposure

Everyone is exposed to very low levels of mercury in their environment, in air (since metallic mercury is slightly volatile), in water or food, and even in the mercury-amalgam dental fillings that might be in a person's teeth (ATSRD 1999). Bacteria easily convert inorganic mercury in the environment into methylmercury, which is the most common form of organic mercury and is the primary form that is found in fish (EFSA 2012). Methylmercury biomagnifies up the food chain through consumption of prey organisms. Fish at the top of the aquatic food chain, such as largemouth or smallmouth bass, can biomagnify methylmercury 10,000 to 100,000 times greater than the surrounding water concentrations (WDOH 2003). Thus, ingestion of contaminated fish can be a significant source of exposure to mercury (ATSRD 1999).

Non-Cancer Toxicity

Multiple cases of accidental methylmercury poisoning have occurred, and the resulting case studies have provided information regarding the toxicity in humans (ATSDR 1999). Some people who were exposed to large amounts of methylmercury in these poisoning episodes developed permanent damage to the brain and kidneys, and deaths (attributed primarily to central nervous system toxicity) were also reported (ATSDR 1999). The nervous system is very sensitive to methylmercury, and the prenatal period is particularly sensitive to effects from exposure (ATSDR 1999).

Long-term oral exposure to high levels of methylmercury in study animals is associated with damage to the kidneys, stomach, and large intestine; changes in blood pressure and heart rate; adverse effects on the developing fetus, sperm, and male reproductive organs; and increases in the numbers of spontaneous abortions and stillbirths (ATSDR 1999). Animal studies indicate that the nervous system is more sensitive to methylmercury toxicity than are other organs in the body (ATSDR 1999). Animal studies provide evidence of damage to the nervous system from exposure

to methylmercury during development, and evidence suggests that the effects worsen with age, even after the exposure stops (ATSDR 1999).

When a pregnant woman is exposed to methylmercury, it is passed through the placenta to the developing fetus and can accumulate in the unborn baby's blood to a concentration higher than that in the mother; it also passes into breastmilk and can continue to affect the developing infant following birth (ATSDR 1999). In critical periods of development before they are born, and in the early months after birth, children and fetuses are particularly sensitive to the effects of methylmercury on the nervous system (ATSDR 1999). With low levels of exposure, some effects might not be readily apparent, such as small decreases in IQ or effects on the brain that can only be revealed through very sensitive neuropsychological testing (ATSDR 1999). With higher exposures, the effects might be more serious but delayed, displayed later as slowed developmental milestones (e.g., age of first walking and talking), or perhaps with more severe effects, such as brain damage with mental retardation, incoordination, and paralysis (ATSDR 1999). With very high levels (such as in the poisoning events mentioned previously), the effects can include eventual blindness, involuntary muscle contractions and seizures, muscle weakness, and inability to speak (ATSDR 1999).

The following toxicity information is based on the EPA (2019c) IRIS Summary data for Methylmercury. EPA (2019c) identifies three relevant human longitudinal (epidemiological) developmental studies associated with exposure to methylmercury contamination in fish: (1) Seychelles Islands study (2) Faroe Islands study and (3) New Zealand study. In each study, exposed infants and children were tested on a number of standardized neuropsychological endpoints (EPA 2019c). Two of the studies (Faroe Islands study and New Zealand study) showed methylmercury exposure associated with poor neurodevelopment functional outcomes, such as dysfunctions in language, attention and memory (EPA 2019c).

The primary study selected by EPA was the Faroe Islands study, which obtained data from about 900 mother-infant pairs (EPA 2019c). Cord-blood mercury, maternal-hair mercury (at the time of childbirth), and child-hair mercury (at 12 months and 7 years of age) were measured, and, at 7 years of age, the children were tested on a variety of tests to evaluate specific behavioral, neurophysiological, and neuropsychological domains (ATSDR 1999). Mercury-related neuropsychological dysfunctions were most pronounced in the domains of language, attention, and memory, and to a lesser extent in visuospatial and motor functions (ATSDR 1999).

Carcinogenicity

EPA has classified methylmercury as a "possible human carcinogen" based on inadequate data in humans and limited evidence of carcinogenicity in animal studies (EPA 2019c). Analysis of human case study data obtained from accidental methylmercury poisoning events does not conclusively indicate any increased cancer incidence among the exposed populations (EPA 2019c). The EPA (2019c) IRIS Summary listed multiple animal studies (that reported methylmercury might be carcinogenic; however, no carcinogenic toxicity value has been derived for methylmercury.

4.0 LEAD

Description

Lead (chemical symbol, Pb) is a metallic element that occurs naturally, sometimes in concentrated ore deposits; it does not degrade but can exist in various chemical forms (ATSDR 2019). Lead is soft and malleable, has a relatively low melting point, and is denser (heavier) than most common materials. Due to these properties and the ease with which it is removed from its ores, it has found many uses since prehistoric times (ATSDR 2019).

Exposure

Lead is ubiquitous in our environment due to its many uses, but perhaps mainly due to the former use in leaded gasoline, which was phased out and eventually banned in 1995 due to the growing understanding of the toxic effects of low-level lead exposure (ATSDR 2019). Lead is not very mobile in the environment and tends to bind to soil and sediment; therefore, it is commonly found in the soil near roadways, around older homes (from chips of old lead-based paint), in old orchards (where lead-containing pesticides were commonly used), and near mining, industrial, or waste sites (ATSDR 2019). For most adults (excluding occupational exposures) exposure occurs primarily from lead in ambient air, drinking water (particularly when lead pipes or lead-soldered pipes are used), food, and soil/dust (ATSDR 2019). Adult occupational exposure in some cases can be significant, and some types of hobbies (such as working with stained glass or making lead fishing weights) can present an important source of exposure (ATSDR 2019). The primary source of exposure for children is ingestion of lead-contaminated soil/dust, especially in older homes containing lead-based paints (ATSDR 2019).

The current lead concentrations in food are generally low (having decreased significantly with the reduced use of lead solder in cans) (ATSDR 2019). The uptake of lead in animals, just as in humans, can occur due to inhalation of contaminated air and the ingestion of contaminated

food and soil/sediment; however, it is not biomagnified in the food chain (ATSDR 2019). Fresh-water fish in lead-contaminated water can accumulate lead in their blood and tissues (particularly in the gills, liver, kidney, and bone) (WDOH 2011). According to WDOH (2007), lead levels in whole fish samples from the Spokane River are "very high" compared to lead in fish from other parts of the state.

Non-Cancer Toxicity

Lead had been known as a toxic substance for over 2000 years, but in the past few decades, there has been growing awareness of the adverse effects associated with low-level exposures, particularly in children (ATSDR 2019). Discussion of toxic levels in lead exposure deals with blood lead levels (PbB) rather than dose levels. Environmental exposure to lead occurs continuously throughout life, and lead can be retained in the body for decades (primarily stored in the bones), and it can be eliminated relatively rapidly from the blood (ATSDR 2019). Therefore, a PbB measurement reflects not only the recent exposure history, but also the cumulative body burden in the bone (ATSDR 2019). Evaluations of PbB have focused on a target level of ≤ 10 µg/dL (micrograms of Pb per deciliter of blood), with that being the accepted threshold for potential adverse effects (ATSDR 2019). The current Center for Disease Control (CDC) reference value (updated in 2012), however, is 5 µg/dL, and it is currently accepted that effects for the most studied endpoints do occur at that level (CDC 2019).

According to ATSDR (2019) there exists an enormous amount of literature studying the toxic effects of lead exposure in adults and in children; therefore, reliance on animal study data (also abundant) is not necessary for lead. Adverse health effects from lead exposure have been observed in every organ system and over a wide PbB range (≤ 5 µg/dL to > 50 µg/dL) (ATSDR 2019). Numerous health outcomes of lead exposure have been studied extensively, including neurological, renal, cardiovascular, hematological, immunological, reproductive, and developmental effects (ATSDR 2019). The following information of these health outcomes was provided by ATSDR (2019) and is summarized below from the ATSDR (2019) document:

- Neurological

Cognitive deficits in children (occurring at ≤ 5 µg/dL) are the best substantiated effects from lead exposure. Infants absorb lead from the mother *in utero* and can absorb additional lead from their mother through ingestion of breast milk. As they grow, the hand-to-mouth behavior of young children makes them more susceptible to elevated lead intake through ingestion of soil/dust and (in older homes) chips of paint containing lead. According to ATSDR (2019), documented neurological effects in children include decrements in cognitive function (learning and memory), altered behavior and mood (attention, hyperactivity, impulsivity, irritably,

delinquency), and altered neuromotor and neurosensory function (visual-motor integration, dexterity, postural sway, changes in hearing and visual thresholds). These effects are associated with PbB in the range of ≤ 5 µg/dL to > 50 µg/dL, with the decrements in cognitive function increasing with PbB level. Numerous studies have provided evidence for effects at PbB ≤ 5 µg/dL. Similar neurological effects are also observed in adults (associated with PbB ranging from ≤ 10 µg/dL to > 50 µg/dL), including peripheral neuropathy, psychiatric symptoms (depression, panic disorders, anxiety, hostility, confusion, anger, and schizophrenia), and changes in regional brain volumes and neurochemistry. It is not clear if these effects are related to adulthood exposure or resulted from earlier developmental exposure (during the prenatal and childhood periods).

- Renal

Adverse renal (kidney) effects at lower levels include enzymuria, proteinuria, impaired transport of organic anions and glucose, and depressed glomerular filtration rate (GFR); at higher PbB (> 30 µg/dL), proximal tubular nephropathy, glomerular sclerosis, interstitial fibrosis, and tubular necrosis. Renal damage can subsequently produce increased lead body burden due to decreased ability to eliminate lead from the blood (reverse causality).

- Cardiovascular

Effects on adult blood pressure is the most-studied cardiovascular outcome. Studies indicate increased systolic and diastolic blood pressure occurring at PbB ≤ 5 µg/dL. Other reported cardiovascular effects include increased risk of hypertension and heart disease, atherosclerosis, altered cardiac conduction, cardiac disease, and increased mortality due to cardiovascular disease.

- Hematological

Lead toxicity to the hematological (blood) system has been associated with decreased blood hemoglobin at PbB ≤ 10 µg/dL; however, the reported decrease at that level was small and might not be of biological significance. As PbB increases, further decreases in blood hemoglobin and loss of erythrocytes result in anemia.

- Immunological

Effects to the immune systems of children and adults (changes in humoral and cell-mediated immunity) have been observed in populations with average PbB < 10 µg/dL. These effects are supported by the results from animal studies, where similar changes lead to sensitization, autoimmunity, and inflammation.

- Reproductive

In males, observed effects of lead (observed from studies of PbB ranging from ≤10 to >50 µg/dL) include damage to the sperm, possible alterations in serum levels of reproductive hormones, decreased fertility, and histopathological changes to the testes, with severity of effects increasing with PbB. In females (PbB ≤ 10 µg/dL), studies provide some evidence of alterations in serum reproductive hormone levels, decreased fertility, increased spontaneous abortion, increased preterm birth, and decreased age at onset of menopause; however, these results are inconsistent, with several studies reporting no association with PbB.

- Developmental (Excluding Neurological Effects)

Some studies provide evidence (associated with PbB ≤ 10 µg/dL) of decreased birth size, decreased child growth, and delayed onset of puberty in males and females. For outcomes other than decreased birth weight (and the neurological effects discussed previously), study results for developmental effects are inconsistent, and some studies show no association.

- Other

Additional effects have been reported in some studies; however, results are inconsistent, and insufficient data are available for development of dose-response relationships: respiratory effects, endocrine effects (excluding reproductive hormones), hepatic (liver) effects, musculoskeletal (bone) effects, gastrointestinal effects, body weight effects, and ocular effects (excluding neurological).

Due to the unique nature of lead toxicity, EPA concluded that it is inappropriate to develop a standard non-cancer toxicity value for lead exposure (EPA 2019d). Instead, EPA has developed two detailed models for the evaluation of potential hazard due to lead exposure: (1) the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children; and (2) the Adult Lead model, for the evaluation of lead exposure to adults, especially pregnant women (EPA 2019d).

Carcinogenicity

Epidemiological studies have provided limited but inconsistent evidence of lead carcinogenicity (ATSDR 2019). At PbB ≤ 10 µg/dL, increased risks were reported for all cancers and lung cancer; at > 10 µg/dL, increased risks were observed for all cancer, respiratory tract cancer, stomach cancer, intestinal cancer, cancer of the larynx, and glioma (ATSDR 2019).

EPA has classified lead as a "probable human carcinogen," based on sufficient evidence in animals (significant increases in renal tumors with dietary exposure to several soluble lead salts); evidence in humans was considered inadequate (EPA 2019d). EPA has not derived a cancer-related toxicity value for lead (EPA 2019d).

5.0 ZINC

Description

Zinc (chemical symbol, Zn) is a naturally occurring metal, one of the most common elements in the Earth's crust, and is present in air, soil, and water (ATSDR 2005). Metallic zinc has many uses in industry, such as in coating steel and iron to prevent rust and corrosion (galvanization), as a component in alloys such as brass and bronze and the alloy used to make pennies, and to make dry cell batteries (ATSDR 2005). Zinc combines with other elements to form zinc compounds (e.g., zinc acetate, zinc chloride, zinc oxide, and zinc sulfate), many of which are widely used in industry, for example, as ingredients in white paints, rubber, wood preservation, dyes, smoke bombs, sun block, diaper rash ointment, and deodorants (ATSDR 2005).

Exposure

Zinc is present in all foods and in most drinking water (ATSDR 2005). It is an essential element needed by the human body in small amounts and is commonly found in vitamin supplements (ATSDR 2005). Zinc enters the air, water, and soil as a result of both natural processes and human activities. Most zinc enters the environment as the result of mining, purifying of zinc, lead, and cadmium ores, steel production, coal burning, and burning of wastes (ATSDR 2005). These activities can increase zinc levels in the atmosphere (ATSDR 2005). Waste streams from zinc and other metal manufacturing and zinc chemical industries, domestic wastewater, and run-off from soil containing zinc can discharge zinc into waterways (ATSDR 2005).

Non-Cancer Toxicity

Acute non-cancer health outcomes associated with oral doses of very high levels (~140-560 mg) of zinc (dose = 2-8 mg/kg/day) include gastrointestinal effects such as vomiting, abdominal cramps, and diarrhea (ATSDR 2005). Longer-term exposure to lower (but elevated) doses (~0.5-2 mg zinc/kg/day) of zinc compounds can produce decreased absorption of copper, leading to early symptoms of copper deficiency, manifesting as decreased erythrocyte number or decreased hematocrit (ATSDR 2005). High doses of zinc have resulted in reductions in leukocyte number and function (ATSDR 2005). Some studies have also found decreases in high-density lipoprotein in humans exposed to increased levels of zinc (ATSDR 2005).

Reduced copper status has been associated with increased zinc intake (EPA 2005). EPA identified four primary studies to support derivation of the non-cancer toxicity value for zinc; the critical effect associated with these studies was decrease in erythrocyte Cu, Zn-superoxide dismutase (ESOD) activity in healthy adult male and female volunteers (EPA 2005). In studies measuring the interactions of excess zinc with copper, there was a consistent decrease in ESOD activity (EPA 2005). Thus, copper status and ESOD activity are considered a sensitive measure of the effects of elevated levels of zinc exposure (EPA 2005).

Carcinogenicity

EPA has categorized zinc as "inadequate information to assess carcinogenic potential", and EPA has not derived a cancer-related toxicity value for zinc (EPA 2005).

6.0 BACTERIA

Description

Bacteria are single-celled organisms that exist all around us and are a normal part of the environmental community in all surface waters (USGS 2019). The specific bacteria of concern in the protection of surface water contact for recreational use is *Escherichia coli* (*E. coli*) (USGS 2019). *E. coli* is a member of the fecal coliform group of bacteria and is commonly found in the gastrointestinal tract and feces of all warm-blooded animals (USGS 2019). The assessment of surface water quality for recreational use includes tests for *E. coli* because the presence of this bacteria in surface water provides direct evidence of fecal contamination in the water (USGS 2019).

Exposure

The primary means of exposure to *E.coli* (and to other disease-causing organisms such as viruses and protozoans that might also be present in recreational surface water) is through ingestion of the surface water while swimming, wading, fishing, or boating (USGS 2019). It is also possible to ingest the organisms through hand-to-mouth behavior or by contamination of food through direct or indirect contact with the surface water (USGS 2019).

Toxicity

Most strains of *E. coli* are harmless, however, some can cause illnesses, such as diarrhea, urinary tract infections, meningitis, septicemia, respiratory illness and pneumonia (USGS 2019). Young children, the elderly, and other people with weakened immune systems are particularly

susceptible to illness from *E. coli* exposure (USGS 2019). The presence of *E. coli* (or fecal coliform bacteria, in general) in water is a strong indicator of contamination from human sewage (such as from a break in a sewage line or improper sewage containment) or animal waste (generally from agricultural runoff), and the human or animal wastes might contain serious disease-causing organisms (USGS 2019).

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